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ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE

DISEASES OF INFANTS AND CHILDREN

JOHN FITCH LANDON, M.D., Editor

LEADING ARTICLES IN THIS NUMBER

Poisoning Due to Petroleum Products.
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Neurologic Disorders in Children. A Study of 282 Hospital Cases.
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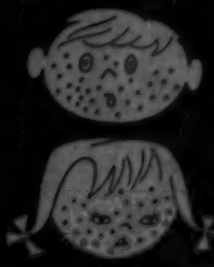

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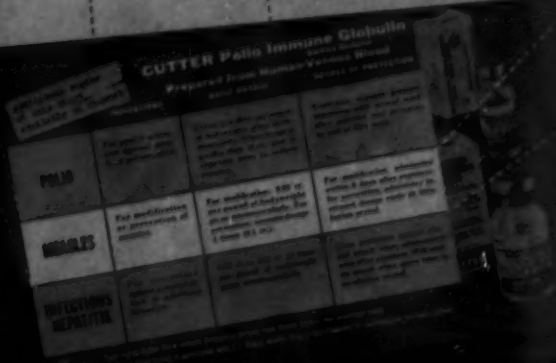

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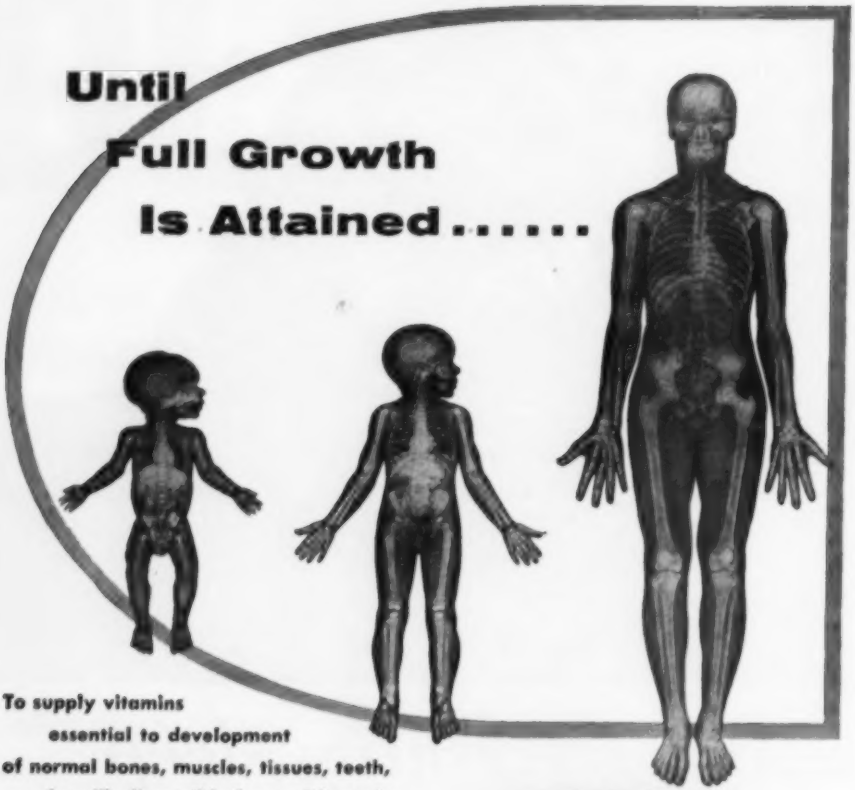

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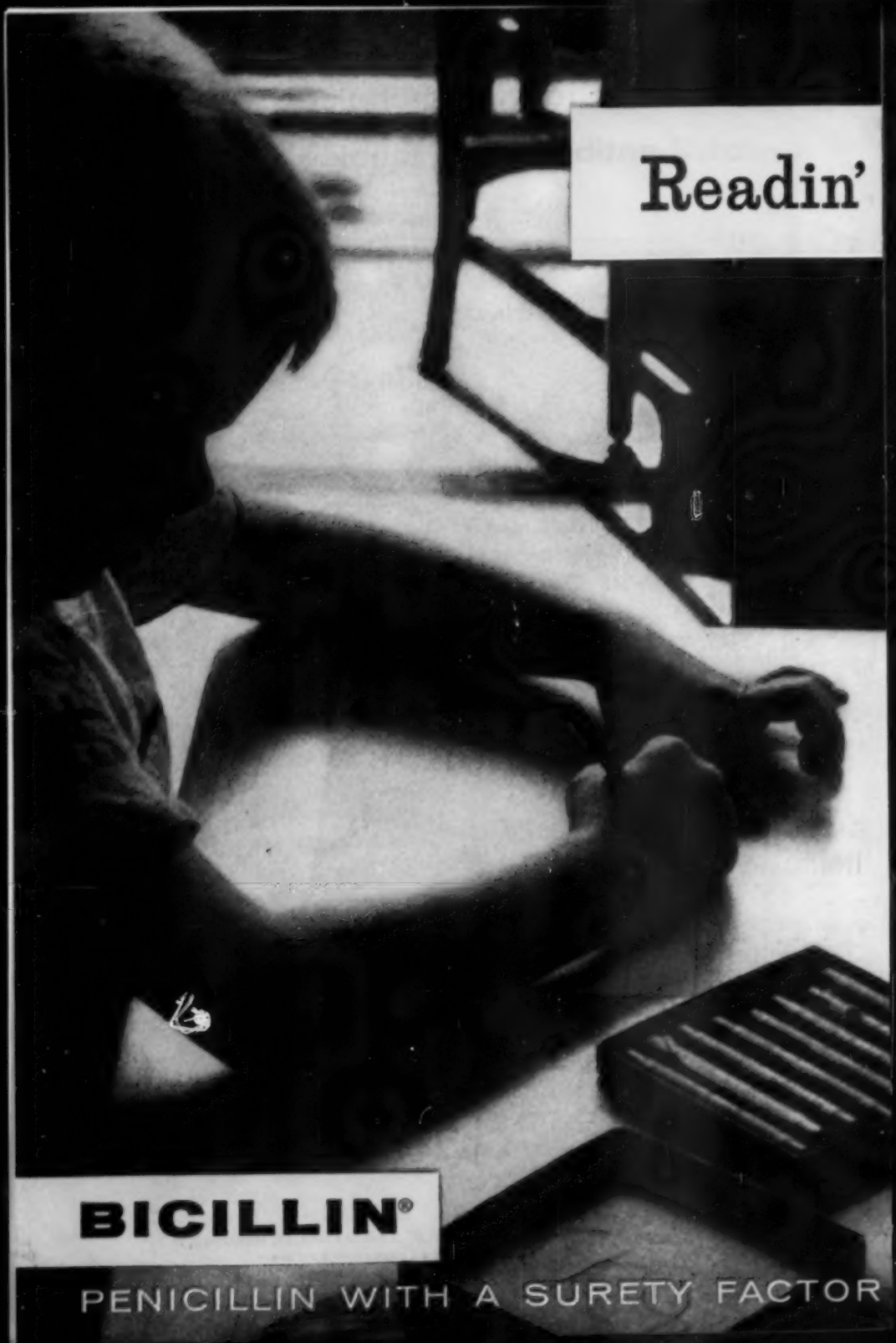
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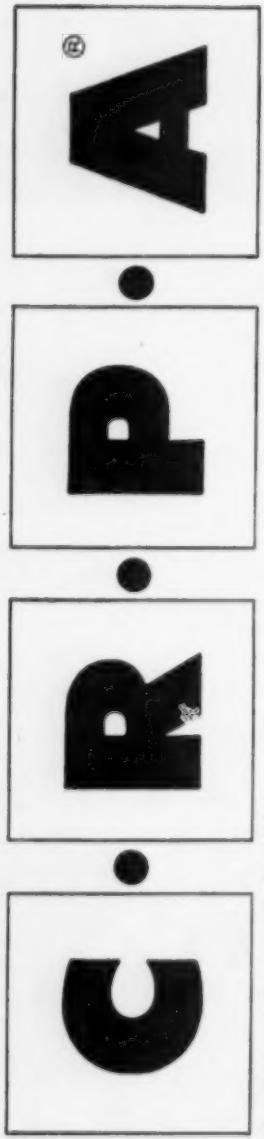

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POISONING DUE TO PETROLEUM PRODUCTS*

PAUL M. THOMPSON, M.D.
Miami, Fla.

Review of the Literature. Kerosene poisoning is in almost all cases due to ingestion of the liquid by children. Inhalation of fumes as an industrial hazard and ingestion by adults are almost nonexistent dangers. According to reports from various hospitals, kerosene poisoning cases exceed those of all other cases of poisoning combined.[14] In spite of this, and although the literature contains much material on the subject, there is but little material on the pathology of kerosene poisoning in most textbooks of pathology. Autopsy material in these cases is relatively scarce.[1, 2, 4, 11]

Mortality from ingestion of kerosene has been found from 6.7 per cent[1] to 11 per cent.[9] Gasoline, which produces similar but more severe clinical and pathologic effects, has a mortality rate of about 28 per cent.[9] Death occurs in 2 to 18 hours.[9] The affected children are usually 1 to 4 years of age and 82 per cent are colored.[4]

Clinical Picture. Clinically, Steiner[16] divides the patients into three categories, depending upon the clinical severity of the poisoning. The first category is that of acute toxicity and depression of the central nervous system with minimal pulmonary changes. The second is that of severe pneumonia, hyperpyrexia and prolonged recovery. In the third type, there is severe pneumonia with evidence of degenerative changes in the myocardium, liver, kidneys and gastrointestinal tract as a result of absorbed kerosene.

The first group of symptoms are due to the irritating effect of

*From the Department of Pathology, Jackson Memorial Hospital and University of Miami, School of Medicine, Miami, Florida.

kerosene upon the upper alimentary tract and the effects of its absorption into the blood stream. There are burning in the mouth and throat, spasm of the glottis, coughing, choking, substernal pain and vomiting. Absorption of kerosene by the alimentary tract or lungs probably leads to the sequential clinical findings of fever, drowsiness and occasional coma, and increased heart and respiratory rates. These patients usually recover. In the second category, the findings of pneumonia become apparent with patchy areas of consolidation detected upon roentgenologic examination. Læsser et al.[7] found 17 of 22 patients who had chest radiographs, showed lung changes although most showed no clinical evidence of lung involvement. Radiographic changes are found as early as one hour after ingestion. In the third category are those patients who show confluent pneumonia by chest radiographs, cardiac dilatation, enlargement of the liver and spleen and urinary changes. Some have been reported to have developed pneumothorax, subcutaneous emphysema and even pneumopericardium.[6, 11, 15]

Laboratory Data. Laboratory studies are of little value. Leukocytosis, with counts up to 20,000, is usually present but is not reliable for diagnosis. Urinary changes, with white blood cells and albumin, are present sometimes.

Roentgenograms. Roentgenograms are of value in demonstrating the pneumonic consolidation. Gershon-Cohen et al.[3] describe it as a bilateral, uniformly disseminated bronchopneumonia which clears within 1 to 3 weeks. These findings are not pathognomonic for the disease process.[4]

Mode of Action. There is still controversy as to the exact mode of action of the kerosene in the production of pathologic manifestations. In experimental animals, Waring[17] with dogs (1933), Læsser et al.[7] with rabbits (1943), Reed et al.[12] with rabbits (1950), and Richardson and Pratt-Thomas[14] with dogs and rabbits (1951), all concluded that aspiration of kerosene into the tracheobronchial tree, aided by gastric regurgitation of kerosene, was the main factor in the production of the pulmonary changes.

On the other hand, Deichmann et al.[1] (1944) believed that rapid gastrointestinal absorption and spread by the blood stream are capable of producing the parenchymal and vascular damage found. Hemorrhages, blood stasis and edema were attributed to blood vessel damage. Pneumonia results from the irritating effect

of kerosene carried to the lungs by the blood. Due to its low volatility, the kerosene is not rapidly excreted by the lungs; therefore, its retention results in a continuous irritation that sets up the inflammatory reaction of pneumonia. Aspiration may be superimposed upon this process. They supported this theory by experiments upon rabbits. Kerosene was given by stomach tube, intravenously, and intraperitoneally. Regardless of the mode of administration of the kerosene, they observed that the lung findings were the same. They also found a marked difference between the oral (28.0 ml./kg.) and intravenous lethal dose (0.18 ml./kg.), indicating that only traces of kerosene are required in the circulation to produce severe pulmonary lesions.

The findings of Richardson and Pratt-Thomas[14], however, seem conclusive in the importance of aspiration in the pathological changes of kerosene poisoning. Using both dogs and rabbits, they gave kerosene by intragastric, intravenous and intratracheal means and by instillation into the small intestine and stomach. They found that it was almost impossible to tell from the histological changes in the lungs which route of administration had been used, although the most severe changes were by the intravenous route. The findings to this point confirmed those of Deichmann et al. However, upon calculating the quantities of kerosene involved, they noted that a child would have to swallow over a pint of kerosene for gastrointestinal absorption to be fatal, if aspiration did not occur. On the other hand, if but one teaspoonful were aspirated, a fatal pneumonitis usually would be expected to result. Therefore, they concluded, that although similar changes may be produced by both modes of action of the kerosene (aspiration and gastrointestinal absorption and spread by the blood stream), by far the most important factor is that of aspiration of kerosene into the tracheobronchial tree and the resultant pneumonia.

Pathology. The pathological findings due to the irritating effect of kerosene are not marked, only slight inflammation of the mouth, pharynx, esophagus and stomach being present. The most prominent findings are in the lungs and have been found in all fatal cases. Nunn and Martin[9], however, suggest that the blood borne effect of kerosene on the central nervous system may be of greater importance.

In humans, autopsied after kerosene poisoning, the lungs are

grossly heavy, red and moist, and have the odor of kerosene. Microscopically, there are hyperemia, edema, hemorrhages, focal interstitial inflammation and an infiltrate of edema, fibrin, polymorphonuclear leukocytes, mononuclear leukocytes and occasional foreign body giant cells in the alveoli. Hyaline membrane formation has been observed.[3] There are necrosis and inflammation of the bronchi with infiltrated polymorphonuclear leukocytes and mononuclears. The adjacent lymph nodes show an inflammatory reaction. The findings are those of an acute hemorrhagic edema and acute bronchopneumonia. They have been found in cases dying as early as two hours after ingestion.[4] The bronchopneumonia later tends to become confluent, and focal atelectasis and emphysema appear. The alveolar walls are weakened by the inflammation and overdistention of the emphysema, and multiple ruptures of these alveoli lead to the infrequent clinically observed pneumothorax and subcutaneous emphysema. Lowery[8] in his case found, in addition to bronchopneumonia, pleural effusion, dilatation of the right heart, dilatation of the stomach and numerous small gastric mucosal ulcerations.

In animals similar changes are found. Waring[17] states that the mononuclears are more pronounced in the alveolar exudate than the polymorphonuclears. With recovery from the disease, there are apparently no pulmonary residua as determined by chest radiographs.[12]

Deichmann et al.[1] found vascular damage in all areas with cloudy swelling of the intima, media and adventitia, perivascular fluid extravasation and perivascular infiltrations of mononuclear leukocytes and lymphocytes. They also observed congestion of the gastrointestinal tract, heart, liver and kidneys. In addition, there were fatty infiltration of the liver lobules, swelling of the von Kupffer cells, swelling of the renal tubular epithelial cells and precipitation of a granular material in the convoluted tubules. The spleen was congested with many phagocytic macrophages and free hemosiderin. The reticulo-endothelial cells were hyperplastic and the follicles large and active.

DISCUSSION OF CASES

Records of cases of kerosene and gasoline poisoning from 3 hospitals were reviewed: Jackson Memorial Hospital; Dade

County Hospital; and Variety Childrens' Hospital. Fifty-five cases of kerosene poisoning, six cases of gasoline poisoning, and one case of lighter fluid poisoniong were found and tabulated (Table 1).

TABLE 1

KEROSENE

Case	Age mos.	Race	Sex	Cough	Vomit	Depress	Lung Phys. exam.	Lung X-ray	Hosp. days	High temp.	White blood count	Gastric Lavage
1	17	C	M	0	0	+	+	+	6	101.6	8,250	0
2	18	C	M	0	0	0	+	+	5	102.4	15,350	0
3	20	C	M	+	+	+	0	0	2	100.2	9,900	+
4	12	W	M	+	0	+	+	+	2	101.2	....	+
5	33	C	M	+	+	0	+	+	10	101.4	7,500	+
6	17	C	M	+	+	0	±	+	2	102.4	9,000	+
7	12	C	M	0	0	+	+	+	5	103	9,500	+
8	18	C	M	+	0	+	+	+	9	104	11,050	0
9	20	C	F	+	0	0	±	0	5	103.4	12,200	0
10	18	W	M	+	0	0	±	0	4	102.4	8,600	0
11	11	C	M	+	+	+	0	0	2	101.4	25,000	0
12	17	C	F	+	+	+	+	+	12	101.6	7,700	0
13	30	C	M	0	0	+	0	0	5	100.6	....	0
14	14	S	F	+	+	0	0	0	3	101	7,700	0
15	34	W	F	+	+	0	0	+	6	102	14,000	+
16	18	C	M	0	0	+	+	—	Exp.	98.2	....	+
17	14	W	F	+	+	0	+	+	2	104.4	15,100	0
18	12	W	F	0	0	+	0	0	5	102	10,000	+
19	14	W	F	+	0	+	+	+	6	102	8,900	+
20	14	W	M	0	+	+	0	+	3	99.6	22,900	0
21	16	C	F	+	+	+	0	+	11	103.2	7,200	+
22	23	W	M	+	0	0	0	0	1	101.1	9,700	+
23	30	C	M	0	0	0	0	+	11	103.6	8,300	+
24	34	W	M	+	+	0	+	+	68	104.6	21,700	0
25	27	C	M	0	+	+	0	±	3	100.2	10,600	0
26	39	C	F	+	+	+	+	+	4	103	8,000	0
27	13	W	F	+	0	0	0	+	1	100.4	....	0
28	14	C	F	+	0	+	0	+	5	102.6	15,700	0
29	12	W	M	+	0	+	0	—	1	100	11,400	0
30	19	C	F	0	+	0	+	+	5	102	6,300	0
31	11	C	M	0	0	0	0	—	2	100.4	6,350	+
32	19	C	M	0	+	+	+	+	11	105.2	15,000	0
33	21	C	F	0	+	+	+	±	4	104.8	17,000	0
34	14	W	M	0	0	0	+	+	5	102.4	17,700	+
35	17	C	M	0	0	0	0	+	4	105	13,150	0
36	19	C	F	0	0	0	0	+	4	103	11,500	0
37	11	C	F	0	+	0	0	+	6	103.2	13,500	+
38	24	C	M	0	+	+	0	0	3	99	9,200	0
39	19	C	F	0	0	0	0	0	3	101.3	16,500	0
40	23	W	M	+	+	+	0	+	11	101.2	13,400	+
41	24	C	F	0	+	0	0	—	4	102	9,550	+
42	9	C	F	0	0	0	0	+	2	101	8,000	0
43	22	C	F	0	+	+	0	0	5	101.4	17,400	0
44	13	C	M	0	0	0	0	+	7	103	14,800	+
45	20	C	F	0	0	0	±	—	3	103.4	10,000	+
46	18	W	F	0	0	0	0	—	1	100.2	....	+

47	14	C	F	0	+	+	0	+	4	105	15,000	0
48	21	W	M	0	0	0	0	0	3	100.4	16,700	0
49	26	W	F	0	+	0	0	+	2	98.8	22,500	+
50	45	W	F	0	0	0	0	—	1	.....	....	0
51	22	W	M	0	0	0	0	+	1	101.6	12,600	+
52	20	W	F	0	0	0	0	+	2	100.8	10,450	0
53	15	W	F	0	+	+	0	+	3	100	10,500	+
54	20	W	M	0	0	0	+	0	1	99.8	5,900	0
55	18	W	F	0	+	+	0	—	Exp.	98.4	5,300	0
GASOLINE												
1	12	W	M	+	+	+	+	+	6	101.4	22,600	+
2	22	W	M	+	+	+	0	+	2	100.3	19,800	+
3	41	W	F	0	0	+	0	—	1	100	....	0
4	17	C	F	0	0	+	0	0	2	101.2	6,400	+
5	13	C	F	0	0	0	0	+	2	100	20,400	0
6	13	C	F	0	0	0	+	+	2	99.2	13,150	0
LIGHTER FLUID												
1	20	C	M	0	0	0	0	+	9	100.2	12,500	0

Kerosene. In the kerosene poisoning group, the ages ranged from 9 to 45 months, averaging 20 months. Of the 55, 32 were colored; 22 were white; and 1 was a Seminole Indian, showing only a slight predominance in the colored race. Twenty-eight were males, and 27 were females, an equal sex distribution.

The volume ingested was usually difficult to ascertain. Estimates, where given, were from 12 to 250 cc. The time following ingestion until the time the patient was first seen also varied, 15 minutes and 72 hours being the extremes; but usually, this also was not recorded.

In their clinical histories, 21 of the 55 patients were recorded as having coughed; 25 were depressed or stuporous; and 25 vomited. In these cases, however, a negative history does not always signify the absence of the symptoms.

The only significant findings on physical examination, besides the mental depression and odor of kerosene, were those of the lungs. Sixteen cases showed definite pulmonary changes with râles and rhonchi. All but one of these were confirmed by chest radiograph or autopsy as showing pneumonic consolidation. Five patients showed questionable lung findings upon physical examination. Two of these showed definite findings on chest radiographs; 2 showed normal chest radiographs, and 1 did not have a roentgenologic examination.

Forty-seven of the 55 patients had chest radiographs. Of these

47, 32 showed definite lung changes upon the chest radiographs (68 per cent); 3 showed questionable changes; and 13 showed no chest abnormalities. Of the 8 not radiographed, 2 expired and both showed autopsy findings of pneumonia.

The duration of hospitalization ranged from 1 to 12 days, averaging 4.4 days, except for a case complicated by atelectasis which was in the hospital for 68 days.

The highest temperature sustained by the patient, excluding the 2 expirations and the complicated cases, ranged from 98.8° to 105° F., averaged 101.7° F., and was usually attained on the first or second hospital day.

The white blood count ranged from 5,900 to 25,500. Of these, 21 were 10,000 or less, and 27 showed a true leukocytosis of over 10,000.

Complications were few. One patient developed atelectasis requiring bronchoscopy and was hospitalized for 68 days. This patient, however, previously had had bronchopneumonia. Another patient developed nephritis. One patient had a lobar type of pneumonia. Incidental coexistent findings were one patient with otitis media and two with ascariasis.

Treatment consisted of supportive measures and, in almost all cases, penicillin. In addition, 18 had gastric lavage, and some received blood, oxygen, or respiratory stimulants.

TABLE 2

	Total	Developing pneumonia	% Relative incidence
Patients who vomited	22	17	77
Patients not vomiting	25	15	68
Patients lavaged	18	15	83
Patients not lavaged	29	19	66
Patients vomiting and/or lavaged	31	24	79
Patients neither vomiting nor lavaged	16	10	63

An attempt was made to correlate vomiting and gastric lavage, and the development of pneumonia in view of the aspiration theory of its development (Table 2). Of 23 patients who vomited, 18 developed definite evidence of pneumonia as determined by chest radiographs or autopsy (relative incidence, 78 per cent). Of the 24 patients who did not vomit, 16 developed pneumonia and 8

did not (relative incidence, 67 per cent). In the other eight cases, evidence of pneumonia was inadequate. These findings indicate only a slight increase in the incidence of pneumonia in those who vomited. It may be contended, however, that more thorough histories in regard to the frequency of vomiting might show a greater relative incidence of pneumonia in those who vomited.

Of the 18 patients whose stomachs were lavaged, 15 developed pneumonia (relative incidence, 83 per cent). Twenty-nine patients were not lavaged. Of these, 19 developed pneumonia (relative incidence, 66 per cent), indicating an even greater but still rather insignificant increase in the incidence of pneumonia in those patients who were lavaged.

Of 32 patients who gave histories of vomiting and/or having had gastric lavage, 25 showed definite evidence of pneumonia and 7 did not (relative incidence, 79 per cent). Of the 15 patients who neither had gastric lavage nor vomited, 9 developed pneumonia (relative incidence, 60 per cent). This again shows a slight, but not markedly significant increase in the relative incidence of pneumonia in those whose stomachs were lavaged and/or who vomited over those who had neither.

The marked similarity in the relative incidence of those who developed pneumonia should also be noted: in those who vomited, 78 per cent; in those lavaged, 83 per cent; and in those who vomited and/or had gastric lavage, 79 per cent. There was only a slightly higher incidence of pneumonia in those who were lavaged.

Similarly, there was a close correlation in those developing pneumonia without vomiting and/or having gastric lavage: in those not vomiting, 67 per cent; in those not lavaged, 66 per cent; and in those neither lavaged nor vomiting, 60 per cent. As would be expected, the incidence of pneumonia was least in the latter, but the difference was slight.

Gasoline. In the gasoline poisoning cases, the number of cases, 6, was inadequate for accurate comparison. However, it was noted that the age range was 12 to 41 months, averaging 20 months. Four were white, and 2 were colored. Three were males, 3 were females.

Symptomatically, 2 were recorded as having coughed; 4 were depressed; and 2 vomited. Three were lavaged. On physical examination, 2 showed positive lung findings. Five had chest radio-

graphs; 4 of these showed the findings of pneumonia. The duration of the hospital stay was 1 to 6 days, averaging 2.5 days. The highest temperature ranged from 99.2° to 101.4° F., averaging 100.4° F. The white blood counts ranged from 6,400 to 22,600. Four of the 5 patients, who had blood counts, showed counts over 13,000.

The number of patients was too small for correlation of pneumonia with vomiting and gastric lavage, but superficially there was no definite relation seen.

Mortality. Two of the patients who ingested kerosene expired, a mortality of 3.6 per cent. They died about 4 and 8 hours after ingestion of the kerosene. Autopsies were performed upon these cases, both of which are reported below.

CASE REPORTS

Case 1. History. M.S., a 15-month-old colored boy, was first seen in the Jackson Memorial Hospital emergency room on May 2, 1954 at 9:00 P.M. about 2 hours after ingestion of an unknown quantity of kerosene. He was in a semistuporous state.

Physical Examination. The patient was a well-developed, well-nourished, lethargic, almost comatose, colored male. His temperature was 98.2° F.; his respiratory rate was 12 per minute, and his heart rate was 110 per minute. Examination of the head, eyes, ears, nose, mouth and throat revealed no abnormalities. Auscultation of the chest revealed bilateral, loud, coarse rhonchi and loud, coarse râles. The abdomen, genitalia and extremities were normal. There was an enlarged left axillary lymph node.

Gastric lavage was performed in the emergency room.

Laboratory Data. The only laboratory study was a hemogram: red blood cells, 4,240,000; hemoglobin, 12.1 gm.%; white blood cells, 9,600, with a differential of 3 stab cells, 60 segmented neutrophils, 30 lymphocytes, 6 monocytes, and 1 eosinophil.

Hospital Course. The patient rapidly developed respiratory failure, became cyanotic, and developed a bloody drainage from the nose and mouth. Respiratory stimulants and artificial respiration were unsuccessful and the patient expired at 11:14 P.M., about 4 hours after ingestion of the kerosene.

Post-mortem Examination. The autopsy was performed 10 hours after death. External examination revealed only a sero-

sanguineous fluid exuding from the nose and mouth and a 1.5 cm. umbilical hernia.

Internal Examination. The brain, meninges and skull showed no abnormalities. In the right pleural cavity there was 50 cc. of cloudy, straw-colored fluid. In the left pleural cavity, a very small amount of a similar fluid was present. The right lung weighed 210 gms. (normal, 72 gms.); the left lung weighed 145 gms. (normal, 65 gms.). Both lungs were a deep reddish-brown in color with only occasional irregular areas of pink, normal-appearing

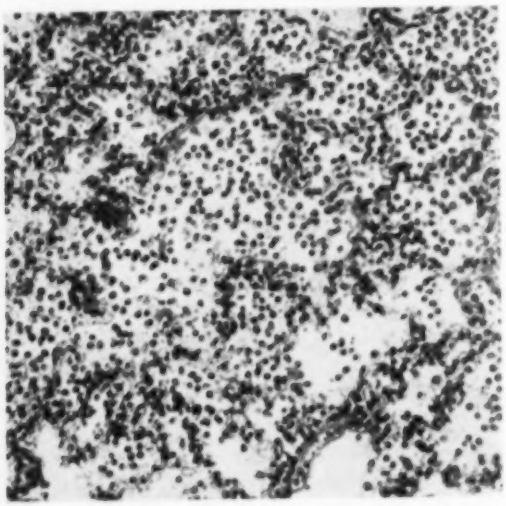

Fig. 1. Case 1. The alveoli contain an exudate of edema fluid, macrophages, lymphocytes and fewer neutrophils. (x133)

lung. The normal crepitation was absent and the lungs had a flabby consistency. The cut surface oozed a cloudy, pink fluid, and a similar fluid was present in the trachea and major bronchi. In addition, there was reddening of the tracheal and bronchial mucosa. The mediastinal lymph nodes were enlarged, pink and soft.

The heart was normal.

The liver, gall-bladder, adrenals, kidneys and bladder showed no abnormalities. The spleen showed accentuation of malpighian corpuscles.

The esophagus was normal. The stomach contained a large amount of partially digested food which had a faint odor of kerosene. The jejunum contained 60 to 80 large round worms (ascaris). The Peyer's patches of the ileum were enlarged and prominent.

Microscopic. The brain showed only mild edema. In the lungs, there were edema of the alveoli and alveolar walls and congestion of the alveolar blood vessels. The edema fluid in the alveoli was frequently vacuolated. In addition, there were foci of pneumonic

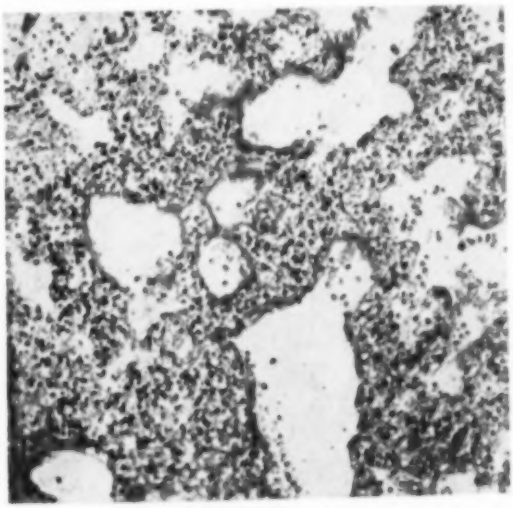

Fig. 2. Case 2. The alveoli and alveolar walls are edematous and infiltrated by mononuclear leukocytes. The edema fluid is frequently vacuolated. The alveoli are lined by hyaline membranes composed of layers of fibrin. (x90)

consolidation around bronchi and bronchioles with infiltrated lymphocytes, neutrophils, eosinophils and macrophages, many with a foamy or vacuolated cytoplasm. The mononuclear leukocytes predominated. There were also larger, more bizarre cells with abundant, eosinophilic cytoplasm and large nuclei, many containing intranuclear or intracytoplasmic, hyaline inclusion bodies. Some of the alveoli were lined by a hyaline membrane composed of laminated fibrin. The bronchioles showed extensive necrosis of

their mucosa with a surrounding inflammatory reaction blending into that of the pneumonic exudate. In some areas the edema was prominent to the exclusion of the pneumonitis. In these areas the bronchioles were unaffected. Fragments of bronchial mucosa were present in some of the alveoli.

In areas the mucosa of the trachea was edematous, congested and infiltrated by lymphocytes and plasma cells. In these areas the mucosal epithelium was absent and the surface was coated with a thin layer of fibrin.

In the heart there were only foci of degenerating myocardial fibers with loss of striations and coagulation of the cytoplasm. The sections of liver, gallbladder, adrenals, bladder, pancreas, thymus, esophagus and stomach showed no abnormalities. The lymphoid follicles of the intestinal submucosa, the spleen and the lymph nodes were large and active. In addition, there were acute inflammatory changes in the lymph nodes with edema and infiltrated polymorphonuclear leukocytes and passive congestion and hemorrhages of the spleen. There were only mild parenchymatous degenerative changes in the kidneys. The bone marrow was slightly hyperactive.

Final Diagnosis. The final pathological diagnosis was: (1) Bronchopneumonia, aspiration type; (2) Intestinal ascariasis; (3) Lymphoid hyperplasia of intestine, spleen and lymph nodes; (4) Subacute mesenteric lymphadenitis.

Case 2. History. D.C., an 18-month-old white girl, drank an unknown quantity of kerosene and a degreasing agent, "Gunk", and was taken immediately to Variety Childrens' Hospital. On the way to the hospital she vomited, began having respiratory difficulty and became unresponsive.

Physical Examination. Upon arrival at the hospital at 2:21 P.M. the patient appeared as an unresponsive white girl with a faint odor of kerosene or mineral spirits about her. Her eyes were rolled back. Her reflexes were hypoactive. The remainder of the examination, including the lungs, was normal.

Laboratory Data. The hemogram was: 4,080,000 red blood cells with 11.3 gms. % hemoglobin; 5,300 white blood cells, with a differential count of 2 stab cells, 6 segmented neutrophils, and 92 lymphocytes. The urinalysis was: pH of 5; specific gravity of 1.018; albumin, 2+; sugar and acetone, negative; 0-1 white blood cells, rare red blood cells, a few epithelial cells, and a few

hypaline to finely granular casts per high power field. The CO_2 capacity was 28 volumes %. There was hemolysis of red blood cells in the CO_2 capacity specimen.

Hospital Course. Because of respiratory distress, she was placed in oxygen and given caffeine sodium benzoate intramuscularly; however, tachypnea, tachycardia and cyanosis continued. Methylene blue, 2 cc. intravenously, was given to combat methemoglobinemia. In addition, intravenous fluids, antihistamines, blood and digitoxin were given.

The patient expired at 9:40 P.M., 7½ hours after admission.

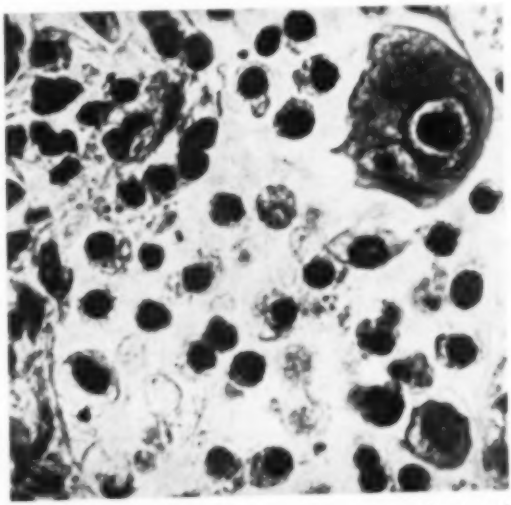

Fig. 3. Case 1. The alveoli contain mononuclears and occasional large bizarre cells which contain densely staining intranuclear inclusion bodies. (x755)

Post-mortem Examination. The autopsy was performed 11 hours after death. External examination revealed no significant abnormalities.

Internal Examination. The brain, meninges and skull had no abnormalities except for congestion of the cerebral veins.

Each of the pleural cavities contained about 15 cc. of a reddish-yellow fluid. The lungs weighed 165 and 155 gms. (normal, 72 and 65 gms.). They were a deep purple in color and had a firm airless consistency. The cut surface was deep red and oozed blood.

The pericardial sac contained about 10 cc. of clear, straw-colored fluid. The heart was normal. The thymus contained an area of ecchymosis.

The peritoneal cavity contained a very small amount of fluid. The liver, gallbladder, spleen, adrenals, kidneys, bladder uterus, tubes, ovaries, pancreas and alimentary tract were normal. The stomach contained a light yellow, viscid, semisolid material.

Microscopic. The brain was normal. In the lungs there was edema of the alveoli and alveolar walls. In addition, there was a pneumonic consolidation of many areas by an exudate composed of edema fluid, polymorphonuclear leukocytes, foamy macrophages, lymphocytes, red blood cells and fibrin in the alveoli. The mononuclear cells and red blood cells predominated. The fibrin frequently formed hyaline membranes lining the alveoli. In some areas there was desquamation of the bronchiolar epithelium.

In the heart there were areas of myocardial degeneration with loss of the striations and vacuolization of the cytoplasm. In the kidneys there was mild parenchymatous degeneration. There were no abnormalities of the thymus, adrenals, spleen, pancreas or bone marrow. In the sections of lymph nodes and intestine there was lymphoid hyperplasia. The stomach and esophagus were normal.

Final Diagnosis. The final pathological diagnosis was: (1) Bronchopneumonia, aspiration type; (2) Edema and congestion of lungs; (3) Lymphoid hyperplasia of intestine and lymph nodes.

DISCUSSION

Grossly, both patients had a small amount of a serous or serosanguineous pleural effusion. The lungs were heavy, moist and red. Microscopically, there were a hemorrhagic edema, foci of pneumonic consolidation with a predominance of mononuclear leukocytes, the formation of hyaline membranes lining the alveoli, and necrosis and desquamation of the bronchial mucosa. In addition, in one case there were intranuclear and intracytoplasmic inclusion bodies. These inclusion bodies, however, may be entirely unrelated to the kerosene aspiration. In the heart and kidneys there were parenchymatous degenerative changes. The main diagnosis was one of a peribronchial and peribronchiolar type of pneumonia typical of aspiration and characterized by a mononuclear leukocytic infiltration and hyaline membrane formation.

SUMMARY

1. The clinical picture, mode of action and pathologic findings of poisoning by petroleum products are reviewed. The majority of the experimental studies favor that of aspiration of kerosene or gasoline into the tracheobronchial tree.

2. Fifty-five cases of kerosene poisoning, 6 cases of gasoline poisoning, and 1 case of lighter fluid poisoning are summarized. In the kerosene cases, 69 per cent developed pneumonia as detected by radiographs or autopsies.

3. In view of the aspiration factor in the development of pneumonia, vomiting and gastric lavage and the development of pneumonia were compared. The most significant finding was that the incidence of pneumonia in those who vomited and/or had gastric lavage was 19 per cent greater than in those who neither vomited nor were lavaged (79 per cent and 60 per cent). In addition, the greatest relative incidence of pneumonia was in those patients who were lavaged (83 per cent). However, it is felt that on the whole the differences are not marked enough to be of major significance.

4. Case reports with autopsy findings are presented on the two patients who expired following kerosene ingestion. The prominent finding in each was an aspiration type of bronchopneumonia characterized by a mononuclear leukocytic infiltration and the formation of hyaline membranes.

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MULTIPLE SCLEROSIS IN CHILDREN. Presse méd., 62:563-564, April 14, 1954). Multiple sclerosis is rare in children, but some true pediatric cases have been reported. The authors maintain that this diagnosis must be made very carefully, in view of the similarity of the symptoms of multiple sclerosis to those of hereditary syphilis, hereditary cerebellar ataxia, Leyden's ataxia, and some of the leukoencephalitic diseases formerly called "acute disseminated encephalomyelitis." One well-tabulated case is presented in which there was no doubt that the disease that began in a boy of 7 and was followed by the authors for 12 years was actually multiple sclerosis. Since the differential diagnosis of multiple sclerosis in children depends principally on noting the manner of evolution of their disease, a long period of investigation is essential. Multiple sclerosis in children often occurs in atypical forms, though the disease process can resemble that seen in young adults. Study of this disease in children is necessarily limited to the chronic forms.—*J.A.M.A.*

THE PEPTIC ULCER PROBLEM IN INFANCY AND CHILDHOOD. (Journal-Lancet, 74:189-191, May 1954). Tudor observed peptic ulcer in 14 children, ranging in age from 5 days to 15 years, and duodenitis in 7 children from 6 to 13 years of age. A review of the literature disclosed that, including Tudor's own cases, a total of 481 cases of peptic ulcer in children have been reported. In the newborn and in infants the mortality of peptic ulcer is 90%. The author's cases included two infants, 5 days and 23 days old, respectively; both died. Ulcers in children beyond infancy respond to a bland diet and an effective antacid such as calcium carbonate. The author believes that peptic ulcer is common during childhood and that infants with cyanosis and abdominal distention and older children with recurrent abdominal pain should be subjected to a thorough radiological examination of the stomach and duodenum to rule out this lesion. Duodenitis in children cannot be distinguished from peptic ulcer on a purely clinical basis.—*J.A.M.A.*

NEUROLOGIC DISORDERS IN CHILDREN*

A Study of 282 Hospital Cases

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Pediatric neurology differs from all other branches of pediatrics by virtue of the fact that unlike diseases of any other system of the body, diseases of the nervous system, if they do not result in immediate death, very frequently do not result in complete recovery either. Severe and permanent crippling, physical, mental or both, may follow many of the neurologic conditions encountered in childhood. Various forms of cerebral palsy, various degrees of mental retardation, various types of epilepsy, emotional disturbances, loss of hearing, loss of vision, speech disorders, either isolated or in various combinations, are frequent sequelae in children who "recovered" from their initial neurologic illness. It is, therefore, of considerable importance to the pediatrician to have the proper orientation and perspective in dealing with these cases. This is important not only in treating the acute conditions but also in foreseeing future developments.

Our experience in one of the largest, if not the largest, pediatric neurology services in the U.S.A. afforded us the opportunity to observe the incidence of the various types of neurologic disorders in children as well as the frequency and type of complications and sequelae. We could also evaluate the effectiveness of the various modes of therapy, especially those of more recent development, such as antibiotics and neurosurgery. We believe that a report of our experience in this field could be of interest to the physician who is called upon to treat similar cases.

This presentation deals with the neurologic cases studied by us at the Children's Division of the Cook County Hospital of Chicago during a 12-month-period, from June 1, 1953 to June 1, 1954. Obviously, in a report of this nature, one cannot dwell at any

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great length upon any one of the conditions considered. Our object in this paper has been, therefore, to present an overall picture, a sort of bird's-eye view of the entire problem, based upon our personal experience.

It should, of course, be kept in mind that the material presented in this paper consists of hospital cases exclusively. It is, therefore, important to note that the type of case encountered in an outpatient clinic devoted to pediatric neurology is entirely different. While the case material seen in a hospital practice consists mainly of the acute neurologic conditions, the cases encountered in an outpatient clinic consist mainly of the many neurologic sequelae that follow those acute conditions.

TABLE I.

Neurologic Disorders. Distribution of Cases According to Their Etiologic Grouping.

Diagnostic Group	Color		Sex		Outcome		Total No. of Cases
	C	W	M	F	Exp.	Dis.	
Heredodegenerative and metabolic conditions	3	2	3	2	1	4	5
Congenital malformations	22	6	12	16	10	18	28
Birth injuries	25	12	19	18	8	29	37
Meningitis	50	15	34	31	12	53	65
Encephalitis and encephalopathy	29	8	21	16	9	28	37
Craniocerebral trauma	19	6	19	6	1	24	25
Vascular and hematologic conditions	18	5	14	9	10	13	23
Neoplastic conditions	3	3	3	3	2	4	6
Convulsions and mental retardation	30	20	26	24	2	48	50
Neuromuscular disorders	4	2	4	2	4	2	6
Totals	203	79	155	127	59	223	282

Key: C—Colored. W—White. M—Male. F—Female. Exp.—Expired. Dis.—Discharged. This key applies to all tables in this paper.

During the one-year-period surveyed by us, 282 neurologic cases were admitted to our Pediatric Neurology Service. During the same period, 4176 medical cases were admitted to the Children's Division of the Hospital. The neurologic cases, therefore, constituted 6.7 per cent of all medical admissions. It may be of interest to compare these figures with those of another year, namely, 1936. The total admissions during 1936 were 3863, while the number of neurologic cases was 209. They thus constituted 5.4 per cent of all admissions.

In order to simplify our study the cases have been grouped according to a classification based upon etiology (Table 1). The various etiologic groupings have been arranged in a temporal order beginning with preconceptual conditions (i.e., hereditary), followed by prenatal (i.e., congenital malformations), then by paranatal (i.e., birth injuries) and, lastly, by postnatal conditions (inflammation, trauma, vascular and neoplastic). The syndromes of convulsions and mental retardation have been dealt with next, and the neuromuscular conditions have been considered last.

A. *Heredodegenerative and Metabolic Conditions.* This group is represented by five cases (Table 2).

TABLE 2.

Heredodegenerative and Metabolic Conditions

Diagnosis	Color		Sex		Outcome		Total No. of Cases
	C	W	M	F	Exp.	Dis.	
Dystonia musculorum deformans	..	1	1	..	1	..	1
Diffuse cerebral sclerosis of Krabbe	1	..	..	1	..	1	1
Myoclonic epilepsy	..	1	1	..	..	1	1
Gargoylism	1	..	..	1	..	1	1
Hand-Schuller-Christian's disease	1	..	1	..	..	1	1
Totals	3	2	3	2	1	4	5

One was a ten-year-old white boy with *dystonia musculorum deformans.* He entered the hospital in the last stages of his illness, which manifested itself by severe muscle spasms, distorted postures and powerful gross involuntary movements. The symptoms appeared at six years of age and gradually became more severe. As in most cases described in the literature, this patient was also of Jewish descent. He expired on the day following admission.

Another was a three-year-old colored female who had been diagnosed previously at the Dr. J. D. Levinson Research Foundation as a case of *diffuse cerebral sclerosis of Krabbe.* She was admitted on account of a diarrhea, but manifested severe neurologic symptoms, consisting of severe mental retardation, marked rigidity, generalized convulsions, blindness and deafness. These symptoms

progressed and followed a normal development up to six months of age. The child was discharged after the diarrhea cleared up.

The third was a three-month-old Puerto Rican boy with *myoclonic epilepsy* of familial occurrence who was discharged two months later.

Another was a thirteen-month-old colored female, who was admitted to the Cook County Hospital on account of a bronchopneumonia. She was found to be mentally retarded. Her facial expression suggested cretinism. However, complete study did not confirm this diagnosis, nor did she improve on thyroid medication. A diagnosis of *gargoylism* was later made.

The last was a six-year-old colored male, who was admitted because of a pneumonia. He was also found to be mentally retarded and, in addition, had various eye palsies, marked hypogenitalism and a bony defect of the frontoparietal area of the skull. A diagnosis of *Hand-Schuller-Christian's* disease seemed the most probable in this case.

TABLE 3.

Congenital Malformation of the Nervous System

Diagnosis	Color		Sex		Outcome		Total No. of Cases
	C	W	M	F	Exp.	Dis.	
Meningocele	7	2	4	5	4	5	9
Hydrocephalus	10	1	5	6	2	9	11
Arnold Chiari's malformation	4	2	2	4	3	3	6
Microcephaly	..	1	..	1	..	1	1
Hydranencephaly	1	..	1	..	1	..	1
Total	22	6	12	16	10	18	28

B. *Congenital Malformation of the Nervous System.* There were 28 cases in this group, of which 16 were newborn babies, while 12 varied in age from one to fourteen months with the exception of one who was five years old. Table 3 shows the distribution of these cases according to diagnosis, color, sex and outcome.

Of the 9 cases of *meningocele* alone, 5 were situated in the lumbar region, associated with *spina bifida* and 4 were situated in the occipital region associated with *cranium bifidum*. Two of the cases with lumbar meningoceles had severe neurologic involvement

of the lower extremities or sphincers or both and, therefore, were not considered operative cases. Of the other 3, one expired before surgery, one after surgery, while the third was operated upon successfully. Of the 4 cases with *occipital meningoceles*, 2 had, in addition, other deformities, namely, missing fingers, absent radii and clubhands in one, and missing cervical vertebra in the other. Interestingly, one of these cases also had the anomaly of the calvarium known as *cranio lacunia* or *Luckenschädel*, which is usually encountered in association with meningomyelocele or meningoencephalocele. One of the 4 cases was successfully treated surgically, while two expired before surgery was attempted, and one was discharged without surgery.

Of the 6 cases of *Arnold Chiari malformation*, 4 had severe involvement of the lower extremities and sphincters, and 3 of these had, in addition, malformations of the lower extremities. One of the meningoceles was repaired surgically but the patient expired before a suboccipital decompression could be attempted. Two others expired before any surgery could be done, and one was discharged without surgery. The sixth patient was a five-year-old boy who, in addition to hydrocephalus and meningocele, had severe neurologic involvement of the lower extremities and sphincters and club feet. He had been operated upon elsewhere previously. His meningocele was repaired, his club feet were orthopedically corrected and his hydrocephalus was relieved by a suboccipital craniotomy. His admission to Cook County Children's Hospital was on account of decubitus ulcers which healed on the ward.

The case of *microcephaly* was that of a white female who was admitted at fourteen days of age with a head circumference of 11 inches (chest 13 inches). Five months later the circumference of the head was 12½ inches (chest 15½ inches). In spite of this, the child developed at a nearly normal rate and a psychological evaluation at the age of five months showed a developmental quotient of 75-80. However, later she began to lag and, although she was alert and responsive, she did not seem to be making normal or nearly normal progress in the intellectual or even motor area.

The case of *hydranencephaly* was admitted at the age of four weeks on account of a mild craniomegaly, the circumference of the head measuring 15 inches (chest 13 inches). Subdural taps revealed about 30 cc. of xanthochromic fluid on each side, and,

therefore, a diagnosis of subdural hematoma was made. At operation, however, a flood of xanthochromic fluid exuded and the entire skull was seen to be empty as far interiorly as the base. There was no evidence of a falx cerebri. The child expired two weeks after the operation, and autopsy confirmed the diagnosis of hydranencephaly.

C. *Birth Injuries of the Nervous System.* There were 37 cases in this group, and their distribution according to diagnosis, color, sex and outcome is shown in Table 4. Of these, 34 had cerebral birth injuries, 2 had brachial plexus birth injuries, and 1 had a spinal cord birth injury.

TABLE 4.

Birth Injuries of the Nervous System

Diagnosis	Color		Sex		Outcome		Total No. of Cases
	C	W	M	F	Exp.	Dis.	
Cerebral birth injury	23	11	17	17	8	26	34
Brachial plexus birth injury	2	..	1	1	..	2	2
Spinal cord birth injury	..	1	1	..	..	1	1
Total	25	12	19	18	9	29	37

The etiologic factors responsible for the 34 cases of cerebral birth injuries were, in order of frequency, prematurity (in 9), caesarean section (in 5), breech delivery (in 3), twin births (in 3), precipitate labor (in 3), dry labor (in 2), prolonged labor (in 1). In 6 cases no etiologic factor could be determined. In 21 cases there was positive evidence in the history of definite cerebral symptoms in the neonatal period, consisting of twitchings, cyanosis and loss of muscle tone. In 7 there was no such evidence, while, in the remaining 6 cases, the information was not elicited.

Of the 34 cases of cerebral birth injury, 12 were newborn babies who were admitted in the stage of acute manifestations of the injury. Of these, 8 expired while 4 survived. No follow-up is, however, available as to the sequelae of those that survived. The remaining 22 cases, varying in ages between two months and seven years, were admitted in the stage of sequelae following the cerebral birth injury. These sequelae consisted of mental retardation,

cerebral palsy and convulsions, either isolated or in combinations. None of these expired.

The 2 cases with *brachial plexus birth injury* were newborn babies. One of these was an upper arm brachial plexus palsy or Erb's type, while the other was of the distal type of brachial plexus palsy or Klumpke's type. Both recovered.

The patient with the *spinal birth injury* was a two-and-a-half-year-old boy with a *complete transection of the spinal cord* since birth. As is usually the case, the injury followed a difficult breech delivery of an oversized baby, with a birth weight of 3.9 kg. He was admitted because of a recurrent urinary infection which cleared up in a few days.

D. *Meningitis.* There was a total of 65 cases in this group. For convenience sake we have included in this group a case of facial paralysis following a tuberculous otitis media. Table 5 shows the distribution of cases of this group according to their diagnosis, color, sex and outcome.

TABLE 5.

Meningitis

Diagnosis	Color		Sex		Outcome		Total No. of Cases
	C	W	M	F	Exp.	Dis.	
Tuberculous meningitis	15	6	8	13	7	14	21
Purulent meningitis	31	7	23	15	3	35	38
Aseptic meningitis	1	1	1	1	..	2	2
Sequellae following purulent meningitis	2	1	1	2	2	1	3
Facial palsy following T. B. otitis	1	..	1	..	..	1	1
Totals	50	15	34	31	12	53	65

The 21 cases of *tuberculous meningitis* varied in age between six months and eight years with the majority (18 cases) being less than three years old. They were practically all treated with streptomycin intramuscularly only, without intrathecal therapy. They also received PAS orally. The more recent cases received oral isoniazid in addition. Seven of the patients expired, 5 were still on the ward at the time this report was written, and 9 were discharged mostly with various sequelae.

The patient with the peripheral *facial palsy due to chronic tuberculous otitis media* was a four-year-old colored male who recovered on antituberculous therapy, and the facial palsy disappeared entirely.

Of the 38 cases of *purulent meningitis,* 9 did not yield any organism on smear or culture of the cerebrospinal fluid. Of the other 29 cases, H. influenza was recovered in 15, pneumococcus in 8, and both together in another 2 cases. Streptococcus, meningococcus, aerobacter aerogenes and eberthela typhoid were found in one case each.

The therapy, in practically all the 38 cases, consisted of aureomycin or terramycin given intravenously for the first 48 to 72 hours, and continued by the oral route after that. The dose varied between 100 mg. and 250 mg. every six hours, administered intravenously in 100 to 250 ml. of 5 per cent dextrose in water. The initial intravenous dose was frequently doubled and the subsequent oral dose was about the same as the intravenous. Other antibiotics were occasionally used in addition to, or instead of, terramycin or aureomycin. Chloromycetin was used in the aerobacter meningitis and in the typhoid meningitis cases.

Of the total 38 cases, 27 recovered completely and uneventfully, 8 developed complications or sequelae, and 3 expired in the acute stage. The etiologic agent in the 3 that expired was H. influenza in 2 and H. influenza plus pneumococcus in the third. Of the 8 patients that developed complications, 4 were complicated by *subdural effusions,* one of whom had a septic arthritis in addition. The etiologic agent in these 4 cases was H. influenza in one, pneumococcus in one, both together in one, and none found in the fourth. All were treated by repeated subdural taps, but two had to be operated upon later. All 4 eventually recovered without sequelae. Two other patients developed *hydrocephalus,* following pneumococcic meningitis in one, and unidentified purulent meningitis in the other. The first was taken home without operation, while the second was operated upon. Both subsequently showed severe neurologic sequelae in the form of a spastic paralysis. The last 2 of the 8 patients, who did not recover uneventfully, had an aerobacter meningitis and an unidentified purulent meningitis, respectively. Both recovered with severe neurologic sequelae in the form of spastic paralysis and mental retardation.

In addition to the 38 cases with acute purulent meningitis, 3 cases were admitted with *sequelae following purulent meningitis*. Two of these 3 patients were admitted because of *hydrocephalus* following the meningitis. In both of these the etiologic agent of the meningitis was unknown. The mechanism of the *hydrocephalus* was found to be an obstruction below the third ventricle in one patient and an extensive adhesive arachnoiditis in the other. In the first case, a ventriculocisternal shunt or a *Torkildsen* operation was performed, while in the second, a ventriculoperitoneal shunt was established. Both expired. The third case was that of a three-year-old severely mentally retarded colored female with a spastic hemiplegia who was admitted because of severe repeated convul-

TABLE 6.

Encephalitis and Encephalopathy

Diagnosis	Color		Sex		Outcome		Total No. of Cases
	C	W	M	F	Exp.	Dis.	
Primary viral encephalitis	2	..	..	2	1	1	2
Post-infectious encephalitis	3	2	3	2	1	4	5
Acute non-specific encephalitis	9	1	6	4	3	7	10
Post-pertussis-inoculation encephalopathy	2	..	..	2	1	1	2
Toxic encephalopathy	4	1	4	1	2	3	5
Congenital toxoplasmosis	..	1	..	1	1	..	1
Sydenham's chorea	3	1	2	2	..	4	4
Acute cerebellar ataxia	..	1	..	1	..	1	1
Lead encephalopathy	5	..	4	1	..	5	5
Salicylate poisoning	1	..	1	..	..	1	1
Tetanus	..	1	1	..	..	1	1
Totals	29	8	21	16	9	28	37

sions. This child had a streptococcic meningitis at the age of two months from which she recovered with severe sequelae.

The two patients with aseptic meningitis recovered without complications.

E. *Encephalitis and Encephalopathy*. There were 37 cases in this group (Table 6).

Of the entire group, only 2 cases had *primary viral encephalitis*. One was a two-year-old colored female whose blood showed antibodies to both *eastern and western equine encephalitis*. She recovered with severe neurologic sequelae. The other was a case of

encephalitis in a seven-month-old colored female who expired, and in whom post-mortem histological examination revealed evidence of *inclusion body encephalitis*.

There were 5 cases of *post-infectious encephalitis*. Three of them followed measles, 1 followed mumps and one six-week-old baby had an encephalopathy accompanying pertussis. One of the cases of measles encephalitis expired. All the others recovered.

The 10 cases of *acute nonspecific encephalitis* varied in age between eighteen months and nine years. Five followed an upper respiratory infection, while 4 had no prodromal symptoms. Of the 9 cases, 3 expired, 2 recovered with neurologic sequelae, while 4 apparently made a complete recovery.

There were 2 cases of *post-pertussis inoculation encephalopathy*. Both were colored females, one and three years of age, respectively. Both were admitted on account of remote sequelae in the form of convulsions following the acute encephalopathy. One of the two expired in status epilepticus.

The 5 cases of *toxic encephalopathy* varied in age between five and eleven months. Three followed bronchopneumonia and 2 followed severe diarrhea. Two expired, 2 recovered completely, while 1 recovered with neurologic sequelae.

There was one case of *congenital toxoplasmosis*. This was a newborn white female with congenital hydrocephalus, bilateral microphthalmia, congenital cataracts, spasticity of the extremities and xanthochromic cerebrospinal, subdural and ventricular fluids with a very high protein content. The methylene blue dye test was positive in a titer of 1:64 in both mother and child. Cerebral calcifications were, however, not revealed by x-rays. The patient eventually expired.

The 4 cases of *Sydenham's chorea* varied in age between five and ten years. It may be of interest to note that during 1936 there were 29 cases of chorea in this same hospital. This diminished incidence of chorea remains to be explained. It may, perhaps, be accounted for by the early treatment with antibiotics of streptococcal throat infections, assuming that the majority of cases of chorea in this country are rheumatic in origin.

There was one case of *acute cerebellar ataxia* with sudden onset in a fifteen-month-old white female who recovered completely in the course of a few weeks.

The 5 cases of *lead encephalopathy* varied in age between one and five years. The diagnosis was established by roentgenologic evidence of a lead line in the metaphysis of the long bones, by basophilic stippling of the erythrocytes, by the presence of coproporphyrin in the urine and by elevated blood lead levels. They were treated by disodium hydrogen phosphate and more recently by calcium disodium versenate.

A case of *salicylate poisoning* in a one-year-old colored male is included in this group since he was admitted in coma with absent reflexes in the lower extremities. A blood salicylate level of 67 mg. per cent was found and a history of aspirin ingestion was obtained. The patient recovered.

The one case of *tetanus* in a nine-year-old white male is also included in this group. His symptoms appeared fourteen days after an injury by a nail in a popliteal region. The child recovered.

F. *Traumatic Lesions of the Nervous System.* There were 25 cases of craniocerebral trauma varying in age between two weeks and eight years. Twenty-three of them were admitted immediately following the injury, while 2 were admitted because of sequelae following head trauma (Table 7).

TABLE 7.

Craniocerebral Trauma

Diagnosis	Color		Sex		Outcome		Total No. of Cases
	C	W	M	F	Exp.	Dis.	
Linear skull fractures	7	2	7	2	..	9	9
Depressed skull fractures	3	1	3	1	..	4	4
Multiple skull fractures	..	1	1	..	1	..	1
Cerebral concussion	7	..	5	2	..	7	7
Cortical laceration without fracture	1	1	1	1	..	2	2
Remote sequellae following head trauma	1	1	2	..	..	2	2
Totals	19	6	19	6	1	24	25

Of the 23 cases, admitted immediately following the head trauma, 9 had a linear skull fracture, 4 had a depressed skull fracture, 1 had multiple skull fractures, 7 had cerebral concussion and 2 had cortical laceration without fracture. Therapy was conservative in all but the depressed fractures, in which the depression was elevated

surgically. Nineteen of the 23 cases recovered completely, although no follow-up is available, 1 expired, while 3 recovered with sequelae. Of the last 3 cases, one, with a linear skull fracture, developed spastic paralysis, mental retardation and convulsions, another, with a cortical laceration, developed spastic paralysis and convulsions, and the third, with a cerebral concussion, had only convulsions. The only patient of the entire group that expired, was the one with mutiple skull fractures.

Of the two patients who were admitted on account of *remote sequelae following head trauma,* one, a four-year-old white boy, developed *post-traumatic epilepsy* after a head injury incurred a year earlier. The other was a six-year-old white boy who, at the age of one-and-one-half years, sustained a head injury which resulted in a spastic hemiplegia, mental retardation and intractable convulsions. At the age of five years, a hemispherectomy was performed in another hospital with the hope of controlling the intractable convulsions and, possibly, improve the mental capacity and the spasticity. However, postoperatively the child remained in a coma. When, about a year later, he was admitted to the Cook County Children's Hospital, pending institutionalization, the patient was still comatose, completely unresponsive, with marked spasticity bilaterally and had to be fed by gavage. He did not have convulsions, however.

G. *Vascular and Hematologic Conditions.* This group includes 23 cases, and their distribution according to diagnosis, color, sex and outcome is shown in Table 8.

Of the 12 cases of *subdural hematoma,* 2 were admitted on account of remote sequelae following the subdural hematoma while 10 were admitted in the acute stage. These 10 varied in age between six weeks and nine months, with the exception of one, who was three days old. The etiologic factor was unknown in most cases, while in 3 a history of postnatal head trauma was elicited. The diagnosis in all cases was based on positive subdural taps. Two of the 10 were discharged, upon the request of the parents, without the benefit of surgical treatment, 3 expired on the day of admission, before any therapy could be instituted, while 5 were treated surgically. Of these 5, 2 expired after surgery because of a complicating meningitis, while 3 were discharged in good condition. Of the 2 patients that were admitted because of remote

sequelae followng subdural hematoma, one was a twelve-month-old colored male who, at the age of two weeks, was operated upon for a subdural hematoma. Later he failed to develop and when admitted to Cook County Children's Hospital, was in a state of complete decerebrate rigidity. The second was a twenty-one-month-old white female who, three weeks after a head injury, sustained at the age of six months, developed a subdural hematoma. She was operated upon but later failed to develop normally, remained mentally retarded and had repeated convulsions.

TABLE 8.

Vascular and Hematologic Conditions

Diagnosis	Color		Sex		Outcome		Total No. of Cases
	C	W	M	F	Exp.	Dis.	
Subdural hematoma	9	3	6	6	5	7	12
Sickle cell anemia with CNS involvement	4	..	2	2	3	1	4
Erythroblastosis foetalis	1	1	2	..	1	1	2
Hemorrhagic disease of the newborn	2	..	2	..	1	1	2
Spontaneous subarachnoid hemorrhage	1	..	..	1	..	1	1
Stürge-Weber disease	1	..	1	..	..	1	1
Cavernous sinus thrombosis	..	1	1	..	..	1	1
Totals	18	5	14	9	10	13	23

The 4 patients with *sickle cell anemia with neurologic involvement* were, of course, all colored and their ages varied between three and eleven years. All were admitted during a sickle cell crisis and all showed neurologic manifestations consisting of lethargy, coma, convulsions and bloody cerebrospinal fluid. One recovered while 3 expired and the autopsies of all 3 confirmed the diagnosis of neurologic involvement of sickle cell anemia.

Of the 2 newborn babies with *erythroblastosis foetalis*, one was the result of an ABO incompatibility, and this patient expired at the age of five weeks. The other was the result of an RH incompatibility and this patient recovered from his hemolysis but showed definite evidence of kernicterus consisting of muscular rigidity and opisthotonos. In neither of the 2 patients was an exchange transfusion done.

Of the 2 patients with *hemorrhagic disease of the newborn*, one

had melena neonatorum in addition to evidence of intracranial hemorrhage; he finally recovered. The other had repeated epistaxis in addition to evidence of intracranial hemorrhage; he finally expired.

The patient with *spontaneous subarachnoid hemorrhage* was a two-year-old colored female with clinical evidence of an intracranial hemorrhage consisting of sudden onset of repeated convulsions and coma. This diagnosis was confirmed by a xanthochromic cerebrospinal fluid containing large numbers of crenated erythrocytes. There was no history or any clinical or radiologic evidence of head trauma. She eventually recovered.

The case of *Stürge-Weber disease* was a seventeen-month-old colored male who was admitted with a history of repeated right-sided convulsions of recent onset. He had a large hemangioma of the left side of the face. Skull x-rays did not however, reveal any intracranial calcifications, although these may develop at a later age.

The case of *cavernous sinus thrombosis* was that of a seven-year-old white male, who following a mild eye infection, developed a high fever with profound lethargy, convulsions, unilateral exophtholmos, periorbital edema, anisocria and xanthochromic cerebrospinal fluid, with slightly raised protein and fifty leucocytes. He recovered on intensive antibiotic therapy.

H. *Neoplastic Conditions.* This group is made up of 6 cases (Table 9).

Two cases, both colored females, three and six years old, respectively, were admitted with ataxia and clinical, ophthalmologic, radiologic and manometric evidence of increased intracranial pressure. A diagnosis of cerebellar tumor was made, and, following operation, a histologic diagnosis of cerebellar *medulloblastoma* was established. Both expired shortly after the surgical procedure.

One was a two-year-old white male who was admitted for the purpose of radiation therapy of multiple metastatic tumors, notably of the face and head, that were diagnosed histologically as *neuroblastoma.* He was discharged after completion of a course of therapy.

Another one was a one-year-old white male with a tumor of the face that was diagnosed histologically as *neurofibroma of Von Recklinghausen.* The patient's mother and maternal grandfather both suffered from the same condition.

The fifth and sixth cases were those of a seven-year-old white female and one-year-old colored male, respectively. A clinical diagnosis of *brain tumor* was made in each case, but they were not operated. They were taken home and later were lost sight of.

TABLE 9.

Neoplastic Conditions

Diagnosis	Color		Sex		Outcome		Total No. of Cases
	C	W	M	F	Exp.	Dis.	
Cerebellar medulloblastoma	2	..	..	2	2	..	2
Neuroblastoma	..	1	1	..	..	1	1
Neurofibromatosis of Von Recklinghausen	..	1	1	..	..	1	1
Brain tumor (unverified)	1	1	1	1	..	2	2
Totals	3	3	3	3	2	4	6

I. *Convulsions and Mental Retardation.* This large and rather heterogeneous group comprises 50 cases whose subdivisions are shown in Table 10.

Generalized convulsions were the most frequent neurologic manifestations among the cases under review in this paper. However, most of the patients who had convulsions have already been considered under their appropriate etiologic groupings responsible for their convulsions. Therefore, under the present heading are included only those cases of convulsions that have not been considered previously.

Similarly, mental retardation was an occasional sequelae of many of the neurologic conditions already considered. Most of the cases of mental retardation, therefore, have been included already under the etiologic grouping responsible for their retardation. Only those cases that have not been considered previously are included in the present group.

There were 15 cases of *idiopathic epilepsy* varying in age between two years and eight years. These were all mentally normal. Seven were admitted with their first convulsion, while 8 were admitted because of convulsions that had been occurring regularly prior to admission. The diagnosis of idiopathic epilepsy was made by exclusion after a very thorough and complete work-up of each case.

There were 14 cases of *febrile convulsions* varying in the age between eight months and three years. In most cases the convulsions accompanied upper respiratory infections. In 10, the convulsions occurred for the first time, while 4 had a history of previous febrile convulsions. In 7, the convulsions occurred singly, while in 7 they recurred two or more times at short intervals.

TABLE 10.

Convulsions and Mental Retardation

Diagnosis	Color		Sex		Outcome		Total No. of Cases
	C	W	M	F	Exp.	Dis.	
Idiopathic epilepsy	11	4	6	9	..	15	15
Febrile convulsions	10	4	8	6	..	14	14
Hypocalcemic tetany	..	2	2	..	..	2	2
Mental retardation of undetermined etiology	8	5	6	7	1	12	13
Mongolism	1	5	4	2	1	5	6
Totals	30	20	26	24	2	48	50

Two patients had convulsions which were found to be due to *hypocalcemic tetany*. They were a set of twins, two months old, who were admitted a week apart with a first convlusion in each case. They were discharged on vitamin D and oral calcium chloride.

There were 13 cases of *mental retardation of undetermined etiology*, varying in age between seven months and seven years. In 9 of these, convulsions were also present, 2 had congenital heart disease in addition to mental retardation, while in 2 no associated condition was present. One of the 13 expired following a bronchopneumonia.

There were 6 cases of *mongolism* that were admitted because of intercurrent respiratory infections. One expired.

J. Neuromuscular Disorders. There were 6 cases in this category and their diagnoses are shown in Table 11.

One was a case of *amyotonia congenita or Oppenheim's disease.* This was a sixteen-month-old colored female, with no familial history of the disease. She was admitted with a bronchopneumonia and expired. The autopsy confirmed the diagnosis of amyotonia congenita.

There was another colored female of thirteen months with a similar clinical picture but with a family history of two siblings dying in infancy of the same condition. There was also a history of progression in this case. She was admitted three times because of repeated pulmonary infections and expired on her last admission. The autopsy in this case revealed *infantile muscular atrophy* or *Werdnig-Hoffmann's disease.*

TABLE II.

Neurovascular Disorders

Diagnosis	Color		Sex		Outcome		Total No. of Cases
	C	W	M	F	Exp.	Dis.	
Amyotonia congenita (Oppenheim)	1	..	..	1	1	..	1
Infantile muscular atrophy	1	..	..	1	1	..	1
Progressive muscular dystrophy	..	1	1	..	..	1	1
Arthrogryposis multiplex congenita	1	..	1	..	..	1	1
Congenital absence of anterior abdominal musculature	1	1	2	..	2	..	2
Totals	4	2	4	2	4	2	6

There was one case of *progressive muscular dystrophy* in a twelve-year-old white male with a family history of the same condition. No pseudohypertrophy was present, the case being of the *Leyden-Moebius* type of muscular dystrophy. He was admitted on account of a paralytic ileus which developed spontaneously three days previously. He was treated conservatively and recovered from the ileus.

One patient had *arthrogryposis multiplex congenita.* This was an eighteen-month-old colored male who had severe contractures in all four extremities which were present at birth and were associated with muscle atrophy but without any disturbance of sensation. This patient was hospitalized because of an intercurrent infection.

The last 2 patients in this group were affected by a *congenital absence of the anterior abdominal musculature.* One was a newborn colored male and the other a seven-weeks-old white male. As is practically always the case there were associated congenital malformations of the urinary tract, consisting of megaloureters and hydronephrosis in both patients. Both expired.

SUMMARY AND CONCLUSIONS

The survey of cases presented here brings out a number of facts regarding neurologic disorders in children:

1. The bulk of neurologic conditions in children encountered in hospital practice consists of acute disorders. This stands in distinct contrast to the neurologic material encountered in an outpatient clinic in which the material consists mainly of the many neurologic sequelae that follow those same acute conditions.

2. The acute nature of the neurologic conditions in hospitalized children also stands in contrast to the chronic nature of most of the neurologic conditions encountered in hospitalized adults.

3. The most frequent neurologic conditions in children are inflammatory in nature, such as meningitis and encephalitis.

4. Antibiotics have completely changed the outlook in the inflammatory conditions of the central nervous system.

5. Next to acute infections, cerebral birth injuries constitute the largest group of neurologic disturbances in children.

6. No notable advances have been made in recent years in the prevention or treatment of cerebral birth injuries, and these are still the most frequent cause of permanent neurologic damage.

7. Congenital malformations constitute a fairly large group and with the development of neurosurgery the outlook in some of them is changing favorably.

8. Craniocerebral trauma also constitutes a large group, although this may be due to the type of cases in our series which belonged mostly to the lower economic strata.

9. Subdural hematoma in infants is not rare and is amenable to surgical treatment.

10. In colored patients, sickle cell anemia must always be considered among the causes of acute neurologic manifestations.

11. The incidence of Sydenham's chorea has decreased to a remarkable degree during the last two decades.

12. Lead encephalopathy is far from rare as a cause of acute neurologic manifestations in children, and because of the disastrous effects upon mental development of prolonged lead intoxication, this cause of neurologic disease must be looked for carefully in order to be removed.

13. Heredodegenerative conditions of the central nervous system are rather infrequently met with in children.

14. Convulsions are the most frequent neurologic manifestation in children and a very thorough work-up is required in order to evaluate completely their significance in any particular case.

15. Pediatric neurology is an integral part of pediatrics.

CONGENITAL AND FAMILIAL AFIBRINOGENEMIA: BASED ON THREE CASES. (Presse méd., 62:1040-1042, July 7, 1954). Congenital and familial afibrinogenemia is a pseudohemophilic disease that manifests itself in early childhood. Nineteen cases were found in the world literature, and three new cases are reported. They occurred in female siblings, the issue of a marriage between first cousins. The parents were both hypofibrinemic. Two brothers were healthy. The three sisters were aged 8½ years, 2 years, and a few weeks old, respectively, when the diagnosis of afibrinogenemia was made. A fourth sister had died from an uncontrollable umbilical hemorrhage at the age of 2 days, and a fifth was hypofibrinemic. The mother had had three spontaneous abortions. The signs and symptoms of the disease are severe hemorrhages appearing in response to minor insults, and superficial ecchymoses and hematomas. The prognosis is poor, but not dire; only 7 of the 22 patients died before the age of 20. Hematologically speaking, the platelets, capillary resistance, and bleeding time are normal in most cases. There is virtually no limit to the coagulation time, blood from one of the authors' patients having been kept for two weeks with no signs of clotting. The sedimentation rate is extremely slow. The almost total absence of fibrinogen, and hence of fibrin, in the blood was shown by various techniques. The mode of transmission of the gene for afibrinogenemia is unknown. Therapy can be only symptomatic. Whole blood, plasma, and fibrinogen have all been used, but in each instance, the patient's blood soon returns to its former state. The difference between this condition and clinical and latent hypofibrinogenemia seems to be simply quantitative.—*J.A.M.A.*

ITEMS

THE FALLACY OF THE FURRED TONGUE. (Brit. M.J., 2:1146-1147, Nov. 13, 1954). A deep-rooted belief exists in both lay and medicinal minds that a dirty tongue, in a child not suffering from an acute illness, is of significance. Statistical analysis of the state of the tongue in 750 children shows that there is no connection between a furred tongue and the state of the tonsils, that of the teeth, the presence of a free nasal airway, the presence of cervical lymphadenopathy, a poor appetite, or the action of the bowels. Similarly, no association can be shown between the state of the tongue and a variety of abnormalities ranging from tongue-tie to tics and from murmurs to mongolism.—*J.A.M.A.*

THE RHESUS FACTOR IN DEAFNESS. (J. Laryng. & Otol., 68:444-460, July 1954). Cavanagh discusses the Rhesus factor, the pathology, clinical aspects and treatment of erythroblastosis fetalis, and the association of deafness and Rhesus sensitization. He reports studies on three groups of children, two of whom had a history of Rhesus factor sensitization. One group consisted of 118 deaf children. In this last group blood samples of the children and their mothers were studied for Rhesus factor incompatibility, and the details of the first two weeks of life were investigated. This group of 118 deaf children included three with Rhesus factor sensitization. Six cases of deafness were found among the 124 children belonging to the two groups with a history of Rhesus factor sensitization. Otologists and pediatricians should give attention to this possible sequel of Rhesus factor incompatibility.—*J.A.M.A.*

CONGENITAL PYLORIC STENOSIS AS A FACTOR PREDISPOSING TO THE ULCER SYNDROME. (Acta pediat., 43:432-443, Sept. 1954). In order to ascertain whether peptic ulcer and gastritis occur more frequently in persons who previously had had manifestations of congenital pyloric stenosis, Nielsen questioned 95 persons (73 men and 22 women), ranging in age from 25 to 45 years, all of whom had received medical treatment for congenital pyloric stenosis in a children's hospital. A group of 100 hospital patients (77 men and 23 women) of the same age range served as controls. Of the patients who had had pyloric stenosis, 28% had gastritis or peptic ulcer, compared with 12% in the control group.—*J.A.M.A.*

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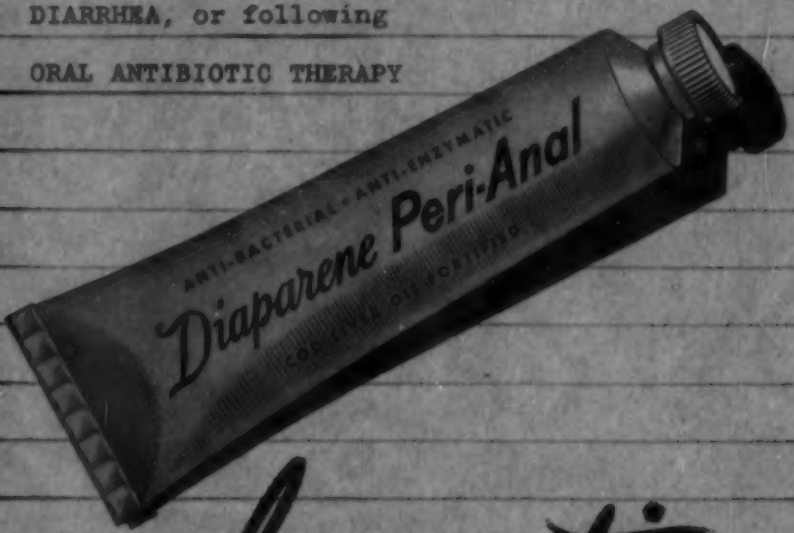

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